WEST Search History

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DATE: Monday, April 10, 2006

Hide?	Set Name	Query	Hit Count
		DB=EPAB,JPAB,DWPI; PLUR=YES; OP=OR	
☐	L18	L17 and (g adj protein adj coupled adj receptor)	4
☐	L17	(corticotropin adj releasing adj factor adj receptor or crf adj receptor or crfr or corticotrophin adj releasing adj factor adj receptor)	243

END OF SEARCH HISTORY

WEST Search History

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DATE: Monday, April 10, 2006

Hide?	Set Name	Query	Hit Count
		DB=PGPB,USPT; PLUR=YES; OP=OR	
☐	L16	L15 and (corticotropin adj releasing adj factor adj receptor or crf adj receptor or crfr)	31
☐	L15	SAWCHENKO	140
☐	L14	L13 and (corticotropin adj releasing adj factor adj receptor or crf adj receptor or crfr)	9
☐	L13	DONALDSON adj CYNTHIA	9
☐	L12	L11 and (corticotropin adj releasing adj factor adj receptor or crf adj receptor or crfr)	15
☐	L11	VALE adj WYLIE	23
☐	L10	L9 and (corticotropin adj releasing adj factor adj receptor or crf adj receptor or crfr)	17
☐	L9	LEWIS adj KATHY	21
☐	L8	L7 and (corticotropin adj releasing adj factor adj receptor or crf adj receptor or crfr)	9
☐	L7	CHEN adj RUOPING	24
☐	L5	L4 and g adj protein	44
☐	L4	L3 and (corticotropin adj releasing adj factor adj receptor or crf adj receptor or crfr)	121
☐	L3	PERRIN	4428

END OF SEARCH HISTORY

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 NEWS  2          "Ask CAS" for self-help around the clock
 NEWS  3  DEC 21  IPC search and display fields enhanced in CA/CAplus with the
                  IPC reform
 NEWS  4  DEC 23  New IPC8 SEARCH, DISPLAY, and SELECT fields in USPATFULL/
                  USPAT2
 NEWS  5  JAN 13  IPC 8 searching in IFIPAT, IFIUDB, and IFICDB
 NEWS  6  JAN 13  New IPC 8 SEARCH, DISPLAY, and SELECT enhancements added to
                  INPADOC
 NEWS  7  JAN 17  Pre-1988 INPI data added to MARPAT
 NEWS  8  JAN 17  IPC 8 in the WPI family of databases including WPIFV
 NEWS  9  JAN 30  Saved answer limit increased
 NEWS 10  JAN 31  Monthly current-awareness alert (SDI) frequency
                  added to TULSA
 NEWS 11  FEB 21  STN AnaVist, Version 1.1, lets you share your STN AnaVist
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 NEWS 12  FEB 22  Status of current WO (PCT) information on STN
 NEWS 13  FEB 22  The IPC thesaurus added to additional patent databases on STN
 NEWS 14  FEB 22  Updates in EPFULL; IPC 8 enhancements added
 NEWS 15  FEB 27  New STN AnaVist pricing effective March 1, 2006
 NEWS 16  FEB 28  MEDLINE/LMEDLINE reload improves functionality
 NEWS 17  FEB 28  TOXCENTER reloaded with enhancements
 NEWS 18  FEB 28  REGISTRY/ZREGISTRY enhanced with more experimental spectral
                  property data
 NEWS 19  MAR 01  INSPEC reloaded and enhanced
 NEWS 20  MAR 03  Updates in PATDPA; addition of IPC 8 data without attributes
 NEWS 21  MAR 08  X.25 communication option no longer available after June 2006
 NEWS 22  MAR 22  EMBASE is now updated on a daily basis
 NEWS 23  APR 03  New IPC 8 fields and IPC thesaurus added to PATDPAFULL
 NEWS 24  APR 03  Bibliographic data updates resume; new IPC 8 fields and IPC
                  thesaurus added in PCTFULL
 NEWS 25  APR 04  STN AnaVist $500 visualization usage credit offered

 NEWS EXPRESS  FEBRUARY 15 CURRENT VERSION FOR WINDOWS IS V8.01a,
               CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
               AND CURRENT DISCOVER FILE IS DATED 19 DECEMBER 2005.
               V8.0 AND V8.01 USERS CAN OBTAIN THE UPGRADE TO V8.01a AT
               http://download.cas.org/express/v8.0-Discover/

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FILE 'HOME' ENTERED AT 09:09:37 ON 10 APR 2006

=> file medline embase biosis caplus
COST IN U.S. DOLLARS — SINCE FILE ENTRY: 0.21 — TOTAL SESSION: 0.21

FULL ESTIMATED COST

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=> s (cortiocotropin(w)releasing(w)factor(w)receptor or crfr) and (GPCR or G(w)protein(w)coupled(w)receptor)
L1 9 (CORTIOCOTROPIN(W) RELEASING(W) FACTOR(W) RECEPTOR OR CRFR) AND (GPCR OR G(W) PROTEIN(W) COUPLED(W) RECEPTOR)

=> dup rem
ENTER L# LIST OR (END):l1
PROCESSING COMPLETED FOR L1
L2 4 DUP REM L1 (5 DUPLICATES REMOVED)

=> dis ibib abs l2 1-4

L2 ANSWER 1 OF 4 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 2005149213 MEDLINE
DOCUMENT NUMBER: PubMed ID: 15781884
TITLE: A novel diuretic hormone receptor in Drosophila: evidence for conservation of CGRP signaling.
AUTHOR: Johnson Erik C; Shafer Orie T; Trigg Jennifer S; Park Jae; Schooley David A; Dow Julian A; Taghert Paul H
CORPORATE SOURCE: Department of Anatomy and Neurobiology, Washington University School of Medicine, St. Louis, MO 63110, USA.
CONTRACT NUMBER: MH067122 (NIMH)
NS27149 (NINDS)
NS56376 (NINDS)
SOURCE: The Journal of experimental biology, (2005 Apr) Vol. 208, No. Pt 7, pp. 1239-46.
Journal code: 0243705. ISSN: 0022-0949.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200506
ENTRY DATE: Entered STN: 20050323
Last Updated on STN: 20050608
Entered Medline: 20050607
AB The Drosophila orphan **G protein-coupled receptor** encoded by CG17415 is related to members of the calcitonin receptor-like receptor (CLR) family. In mammals, signaling from CLR receptors depend on accessory proteins, namely the receptor

activity modifying proteins (RAMPs) and receptor component protein (RCP). We tested the possibility that this Drosophila CLR might also require accessory proteins for proper function and we report that co-expression of the mammalian or Drosophila RCP or mammalian RAMPs permitted neuropeptide diuretic hormone 31 (DH31) signaling from the CG17415 receptor. RAMP subtype expression did not alter the pharmacological profile of CG17415 activation. CG17415 antibodies revealed expression within the principal cells of Malpighian tubules, further implicating DH31 as a ligand for this receptor. Immunostaining in the brain revealed an unexpected convergence of two distinct DH signaling pathways. In both the larval and adult brain, most DH31 receptor-expressing neurons produce the neuropeptide corazonin, and also express the **CRFR**-related receptor CG8422, which is a receptor for the neuropeptide diuretic hormone 44 (DH44). There is extensive convergence of CRF and CGRP signaling within vertebrates and we report a striking parallel in Drosophila involving DH44 (CRF) and DH31 (CGRP). Therefore, it appears that both the molecular details as well as the functional organization of CGRP signaling have been conserved.

L2 ANSWER 2 OF 4 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2001:79677 BIOSIS
DOCUMENT NUMBER: PREV200100079677
TITLE: Corticotropin releasing factor prevents apoptosis induced by camptothecin in Y79 cells.
AUTHOR(S): Radulovic, M. [Reprint author]; Spiess, J.
CORPORATE SOURCE: Max Planck Inst. Exp. Med., Goettingen, Germany
SOURCE: Society for Neuroscience Abstracts, (2000) Vol. 26, No. 1-2, pp. Abstract No.-189.8. print.
Meeting Info.: 30th Annual Meeting of the Society of Neuroscience. New Orleans, LA, USA. November 04-09, 2000. Society for Neuroscience.
ISSN: 0190-5295.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 7 Feb 2001
Last Updated on STN: 12 Feb 2002

AB Corticotropin-releasing factor (CRF) plays a major role in regulating the stress response through the hypothalamic-pituitary-adrenal axis. The action of CRF is exhibited through CRF receptors (**CRFR**) which belong to a **G protein-coupled receptor** superfamily with seven transmembrane domains and are expressed as different subtypes, CRFR1 and CRFR2. We have found that receptors for CRF are frequently expressed in human tumor cell lines originating from different organs. The aim of this study was to examine whether CRF has an effect on survival of Y79 retinoblastoma cells treated by an apoptosis-inducing agent. Both types of CRF receptors are constitutively expressed by the Y79 cell line. CRF (0,1 - 100 nM) was shown to be able to completely reverse cell death induced by a 6h treatment with 0.2 microM camptothecin, a DNA damaging agent used widely in cancer therapy. Cell death was assessed by measurement of lactate dehydrogenase release to a serum-free medium. CRF exerted its pro-survival effect by preventing the apoptotic process induced by camptothecin as determined by measuring activities of caspases 3 and 8. The obtained results indicate a mechanism of a potentially deleterious effect of stress on the outcome of chemotherapy in the treatment of tumors that bear **CRFR**. (Supported by the Max Planck Society)

L2 ANSWER 3 OF 4 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2001:134625 BIOSIS
DOCUMENT NUMBER: PREV200100134625
TITLE: Prevention of complex type glycosylation does not alter the biological activity of CRF receptor 1.
AUTHOR(S): Hofmann, B. A. [Reprint author]; Eckart, K.; van Werven,

L.; Spiess, J.
CORPORATE SOURCE: Max Planck Inst. Exp. Med., Goettingen, Germany
SOURCE: Society for Neuroscience Abstracts, (2000) Vol. 26, No. 1-2, pp. Abstract No.-808.2. print.
Meeting Info.: 30th Annual Meeting of the Society of Neuroscience. New Orleans, LA, USA. November 04-09, 2000. Society for Neuroscience.
ISSN: 0190-5295.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 14 Mar 2001
Last Updated on STN: 15 Feb 2002

AB Corticotropin-releasing factor receptor (CRFR), a G-**protein coupled receptor**, contains an approximately 120 residue extracellular amino terminus. Five to six potential glycosylation sites are located in the N-terminal domain. CRFR carrying complex or high-mannose carbohydrates was identified in the anterior pituitary and the frontal cortex (Grigoriadis, D.E. et al. Endocrinology (1989) 125: 1877-1888). It was the objective of this study to determine whether the carbohydrate moieties of these receptors modulate the ligand recognition or the coupling to effector systems. HEK 293 cells transfected with cDNA coding for rat CRFR1 (rCRFR1) were cultured in absence and presence of kifunensine, an inhibitor of mannosidase I required for the formation of glycoproteins carrying complex carbohydrates. In the presence of this inhibitor, predominantly high mannose type rCRFR1 (rCRFR1-high) was formed as was demonstrated by deglycosylation with EndoHf and Western blot analysis. Under these conditions, no rCRFR1 glycosylated with complex type carbohydrates (rCRFR1-comp) was found. With a scintillation proximity assay based on lectin binding, the binding affinity of h/rCRF was found identical for both receptor species. Similarly, no differences were observed for the affinity of rUcn. Furthermore, the EC 50 values for cAMP accumulation were not changed by kifunensine treatment. It is concluded that the biological activity of rCRFR1 tolerates significant changes of the carbohydrate moieties which may be more important for maturation. (Supported by the Max Planck Society)

L2 ANSWER 4 OF 4 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 96278921 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8662941
TITLE: The genomic structure of the rat corticotropin releasing factor receptor. A member of the class II G **protein-coupled receptors**.
AUTHOR: Tsai-Morris C H; Buczko E; Geng Y; Gamboa-Pinto A; Dufau M L
CORPORATE SOURCE: Section on Molecular Endocrinology, Endocrinology and Reproduction Research Branch, NICHD, National Institutes of Health, Bethesda, Maryland 20892-4510, USA.
SOURCE: The Journal of biological chemistry, (1996 Jun 14) Vol. 271, No. 24, pp. 14519-25.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-U53486; GENBANK-U53487; GENBANK-U53488; GENBANK-U53489; GENBANK-U53490; GENBANK-U53491; GENBANK-U53492; GENBANK-U53493; GENBANK-U53494; GENBANK-U53495; GENBANK-U53496; GENBANK-U53497; GENBANK-U53498; GENBANK-U53499; GENBANK-U53500
ENTRY MONTH: 199608
ENTRY DATE: Entered STN: 19960828
Last Updated on STN: 19960828

Entered Medline: 19960820

AB Isolation and structural characterization of the rat corticotropin releasing factor receptor (**CRFR**) gene was performed to determine the exon/intron organization of the coding region and the potential for splice variants. The **CRFR** gene contains 13 exons and 12 introns, and the positions of the exon/intron junctions are similar to those of other Class II **G protein-coupled receptor** genes including the parathyroid hormone and glucagon receptors. The promoter resides within 593 base pairs of the initiation codon and the major transcriptional start site at nucleotide -238. This domain does not possess a TATA box but contains multiple Sp1 and AP-2 sites upstream and downstream of the major transcriptional start site. Intron junctions were identified in the extracellular, transmembrane (TM), and cytoplasmic (C) domains of the **CRFR**, giving the potential for differential signal transduction by splice variants. **CRFR** cDNAs derived from rat Leydig cell mRNA included the pituitary Form A, which spans exons 1-13, and two splice variants with deletion of exon 3 or exons 7, 11, and 12. An evolutionary link between the intronless TM/C module of the glycoprotein hormone receptors and the intron-containing TM/C module of the **CRFR** is suggested by the common position of the luteinizing hormone receptor Form D alternate acceptor splice site and the **CRFR** intron 12.

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=> FIL STNGUIDE
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                        ENTRY    SESSION
FULL ESTIMATED COST                                     27.95      28.16

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     FILE 'MEDLINE, EMBASE, BIOSIS, CAPLUS' ENTERED AT 09:09:57 ON 10 APR 2006
L1             9 S (CORTIOCOTROPIN(W)RELEASING(W)FACTOR(W)RECEPTOR OR CRFR) AND
L2             4 DUP REM L1 (5 DUPLICATES REMOVED)

     FILE 'STNGUIDE' ENTERED AT 09:13:28 ON 10 APR 2006

=> s (cortiocotropin(w)releasing(w)factor(w)receptor or crfr)
               0 CORTIOCOTROPIN
               0 RELEASING
               7 FACTOR
               5 FACTORS
              11 FACTOR
                 (FACTOR OR FACTORS)
               0 RECEPTOR
               0 CORTIOCOTROPIN(W)RELEASING(W)FACTOR(W)RECEPTOR
               0 CRFR
L3             0 (CORTIOCOTROPIN(W)RELEASING(W)FACTOR(W)RECEPTOR OR CRFR)

=> s (corticocotropin(w)releasing(w)factor(w)receptor or crfr)
               0 CORTICOCOTROPIN
               0 RELEASING
               7 FACTOR
               5 FACTORS
```

```
            11 FACTOR
                 (FACTOR OR FACTORS)
             0 RECEPTOR
             0 CORTICOCOTROPIN(W)RELEASING(W)FACTOR(W)RECEPTOR
             0 CRFR
L4           0 (CORTICOCOTROPIN(W)RELEASING(W)FACTOR(W)RECEPTOR OR CRFR)
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NEWS 4	JAN 13	IPC 8 searching in IFIPAT, IFIUDB, and IFICDB
NEWS 5	JAN 13	New IPC 8 SEARCH, DISPLAY, and SELECT enhancements added to INPADOC
NEWS 6	JAN 17	Pre-1988 INPI data added to MARPAT
NEWS 7	JAN 17	IPC 8 in the WPI family of databases including WPIFV
NEWS 8	JAN 30	Saved answer limit increased
NEWS 9	FEB 21	STN AnaVist, Version 1.1, lets you share your STN AnaVist visualization results
NEWS 10	FEB 22	The IPC thesaurus added to additional patent databases on STN
NEWS 11	FEB 22	Updates in EPFULL; IPC 8 enhancements added
NEWS 12	FEB 27	New STN AnaVist pricing effective March 1, 2006
NEWS 13	FEB 28	MEDLINE/LMEDLINE reload improves functionality
NEWS 14	FEB 28	TOXCENTER reloaded with enhancements
NEWS 15	FEB 28	REGISTRY/ZREGISTRY enhanced with more experimental spectral property data
NEWS 16	MAR 01	INSPEC reloaded and enhanced
NEWS 17	MAR 03	Updates in PATDPA; addition of IPC 8 data without attributes
NEWS 18	MAR 08	X.25 communication option no longer available after June 2006
NEWS 19	MAR 22	EMBASE is now updated on a daily basis
NEWS 20	APR 03	New IPC 8 fields and IPC thesaurus added to PATDPAFULL
NEWS 21	APR 03	Bibliographic data updates resume; new IPC 8 fields and IPC thesaurus added in PCTFULL
NEWS 22	APR 04	STN AnaVist $500 visualization usage credit offered

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                                                           ENTRY    SESSION
FULL ESTIMATED COST                                         0.21       0.21

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or corticotrophin adj releasing adj factor adj receptor)
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The previous command name entered was not recognized by the system.
For a list of commands available to you in the current file, enter
"HELP COMMANDS" at an arrow prompt (=>).

=> s (corticotropin adj releasing adj factor adj receptor or crf adj receptor or
crfr or corticotrophin adj releasing adj factor adj receptor)
L1          146 (CORTICOTROPIN ADJ RELEASING ADJ FACTOR ADJ RECEPTOR OR CRF ADJ
                RECEPTOR OR CRFR OR CORTICOTROPHIN ADJ RELEASING ADJ FACTOR ADJ
                RECEPTOR)

=> dup rem
ENTER L# LIST OR (END):l1
PROCESSING COMPLETED FOR L1
L2           66 DUP REM L1 (80 DUPLICATES REMOVED)

=> dis ibib abs l2 50-66

L2   ANSWER 50 OF 66  BIOSIS  COPYRIGHT (c) 2006 The Thomson Corporation  on
     STN
ACCESSION NUMBER:    2001:134625  BIOSIS
DOCUMENT NUMBER:     PREV200100134625
TITLE:               Prevention of complex type glycosylation does not alter the
                     biological activity of CRF receptor 1.
AUTHOR(S):           Hofmann, B. A. [Reprint author]; Eckart, K.; van Werven,
                     L.; Spiess, J.
CORPORATE SOURCE:    Max Planck Inst. Exp. Med., Goettingen, Germany
SOURCE:              Society for Neuroscience Abstracts, (2000) Vol. 26, No.
                     1-2, pp. Abstract No.-808.2. print.
                     Meeting Info.: 30th Annual Meeting of the Society of
                     Neuroscience. New Orleans, LA, USA. November 04-09, 2000.
                     Society for Neuroscience.
                     ISSN: 0190-5295.
DOCUMENT TYPE:       Conference; (Meeting)
                     Conference; Abstract; (Meeting Abstract)
LANGUAGE:            English
ENTRY DATE:          Entered STN: 14 Mar 2001
                     Last Updated on STN: 15 Feb 2002
AB   Corticotropin-releasing factor receptor (CRFR), a G-protein
     coupled receptor, contains an approximately 120 residue extracellular
     amino terminus.  Five to six potential glycosylation sites are located in
     the N-terminal domain.  CRFR carrying complex or high-mannose
```

carbohydrates was identified in the anterior pituitary and the frontal cortex (Grigoriadis, D.E. et al. Endocrinology (1989) 125: 1877-1888). It was the objective of this study to determine whether the carbohydrate moieties of these receptors modulate the ligand recognition or the coupling to effector systems. HEK 293 cells transfected with cDNA coding for rat CRFR1 (rCRFR1) were cultured in absence and presence of kifunensine, an inhibitor of mannosidase I required for the formation of glycoproteins carrying complex carbohydrates. In the presence of this inhibitor, predominantly high mannose type rCRFR1 (rCRFR1-high) was formed as was demonstrated by deglycosylation with EndoHf and Western blot analysis. Under these conditions, no rCRFR1 glycosylated with complex type carbohydrates (rCRFR1-comp) was found. With a scintillation proximity assay based on lectin binding, the binding affinity of h/rCRF was found identical for both receptor species. Similarly, no differences were observed for the affinity of rUcn. Furthermore, the EC 50 values for cAMP accumulation were not changed by kifunensine treatment. It is concluded that the biological activity of rCRFR1 tolerates significant changes of the carbohydrate moieties which may be more important for maturation. (Supported by the Max Planck Society)

L2 ANSWER 51 OF 66 MEDLINE on STN DUPLICATE 23

ACCESSION NUMBER: 1999296870 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10366634
TITLE: Modulation of learning and anxiety by corticotropin-releasing factor (CRF) and stress: differential roles of CRF receptors 1 and 2.
AUTHOR: Radulovic J; Ruhmann A; Liepold T; Spiess J
CORPORATE SOURCE: Max Planck Institute for Experimental Medicine, Department for Molecular Neuroendocrinology, 37075 Goettingen, Germany.
SOURCE: The Journal of neuroscience : the official journal of the Society for Neuroscience, (1999 Jun 15) Vol. 19, No. 12, pp. 5016-25.
Journal code: 8102140. E-ISSN: 1529-2401.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199906
ENTRY DATE: Entered STN: 19990714
Last Updated on STN: 20010521
Entered Medline: 19990628

AB The differential modulation of learning and anxiety by corticotropin-releasing factor (CRF) through CRF receptor subtypes 1 (CRFR1) and 2 (CRFR2) is demonstrated. As learning paradigm, context- and tone-dependent fear conditioning of the mouse was used. Injection of CRF into the dorsal hippocampus before training enhanced learning through CRFR1 as demonstrated by the finding that this effect was prevented by the local injection of the unselective CRFR antagonist astressin, but not by the CRFR2-specific antagonist antisauvagine-30 (anti-Svg-30). In contrast, injection of CRF into the lateral intermediate septum impaired learning through CRFR2, as demonstrated by the ability of antisauvagine-30 to block this effect. When antisauvagine-30 was injected alone into the lateral intermediate septum, learning was enhanced. Such tonic control of learning was not observed when astressin or antisauvagine-30 was injected into the dorsal hippocampus. Injection of CRF after the training into the dorsal hippocampus and the lateral intermediate septum also enhanced and impaired learning, respectively. Thus, it was indicated that CRF acted on memory consolidation. It was concluded that the observed effects reflected changes of associative learning and not arousal, attention, or motivation. Although a dose of 20 pmol human/rat CRF was sufficient to affect learning significantly, a fivefold higher dose was required to induce anxiety by injection into the septum. Immobilization for 1 hr generated a stress response that included

the induction of anxiety through septal CRFR2 and the subsequent enhancement of learning through hippocampal CRFR1. The involvement of either receptor subtype was demonstrated by region-specific injections of astressin and antisauvagine-30.

L2 ANSWER 52 OF 66 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 2000:70497 BIOSIS
DOCUMENT NUMBER: PREV200000070497
TITLE: High-affinity binding of urocortin and astressin but not CRF to G protein-uncoupled CRFR1.
AUTHOR(S): Ruhmann, Andreas [Reprint author]; Bonk, Ines; Kopke, Andreas K.E.
CORPORATE SOURCE: Radiopharmaceuticals Division, Australian Nuclear Science and Technology, Organisation, Menai, NSW, Australia
SOURCE: Peptides (New York), (Nov., 1999) Vol. 20, No. 11, pp. 1311-1319. print.
CODEN: PPTDD5. ISSN: 0196-9781.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 16 Feb 2000
Last Updated on STN: 3 Jan 2002

AB The structure-activity relationship (SAR) between the recently identified neuropeptide urocortin (Ucn) and corticotropin-releasing factor (CRF) receptor, type 1 (**CRFR** 1), has been investigated. To this end, rat Ucn (rUcn), ovine CRF (oCRF) and chimeric peptides of rUcn and oCRF were synthesized and tested for their binding affinity and potency to stimulate cAMP production in human embryonic kidney (HEK) 293 cells stably transfected with cDNA encoding rat CRFR1 (rCRFR1). In binding studies with (125I-Tyr0)oCRF or (3H-Leu9)rUcn as radioligand, it was observed that rUcn but not oCRF bound in a similar fashion as the CRF antagonist astressin with high affinity to rCRFR1 coupled to G protein or uncoupled from G protein by guanosine 5'-O-(3-thiotriphosphate) (GTPgammaS). Consequently, rUcn was found to exert a significantly lower potency than oCRF to stimulate cAMP accumulation in transfected cells. CD spectroscopic investigations and reverse-phase HPLC (RPHPLC) retention behavior of the peptides suggested a more pronounced amphipatic alpha-helical character of rUcn when compared to oCRF and the chimeric peptides.

L2 ANSWER 53 OF 66 MEDLINE on STN DUPLICATE 24

ACCESSION NUMBER: 1999451236 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10519912
TITLE: Actions of CRF and its analogs.
AUTHOR: Eckart K; Radulovic J; Radulovic M; Jahn O; Blank T; Stiedl O; Spiess J
CORPORATE SOURCE: Max-Planck Institute for Experimental Medicine, Department of Molecular Neuroendocrinology, Hermann-Rein-Str. 3, 37075, Goettingen, Germany.
SOURCE: Current medicinal chemistry, (1999 Nov) Vol. 6, No. 11, pp. 1035-53. Ref: 172
Journal code: 9440157. ISSN: 0929-8673.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199912
ENTRY DATE: Entered STN: 20000113
Last Updated on STN: 20000113
Entered Medline: 19991215

AB Corticotropin-releasing factor (CRF), urocortin, sauvagine and urotensin I form the CRF family. These peptides bind with different affinities to two subtypes of CRF receptor (**CRFR**), CRFR1 and CRFR2. The latter

exists as two splice variants, the neuronal CRFR2a and the peripheral CRFR2b. **CRFR** is a G protein-dependent receptor which acts mainly through Gs enhancing cAMP production. However, CRFR1 expressed in neutrophils of the spleen in response to immunologic stimulation and psychological stress does not seem to function through Gs, as indicated by the inability of CRF to stimulate the cAMP production of CRFR1+ neutrophils. Besides the two receptors, a 37 kD CRF binding protein (CRF-BP) binds several CRF peptides with high affinity. **CRFR** and CRF-BP do not share a common amino acid sequence representing the ligand binding site. In view of the unusually slow offrate of CRF-BP, it is proposed that CRF-BP provides an efficient uptake of free extracellular CRF. Thus, the time of exposure of **CRFR** to CRF or urocortin can be limited. At this time, the fate of the ligand CRF-BP complex is unclear. CRFR1 is not only involved in the hypophyseal stimulation of corticotropin release, but hippocampal CRFR1 mediates enhancement of stress-induced learning. CRFR1 may also be involved in basic anxiety. In contrast, at least in the mouse, CRFR2 of the lateral intermediate septum mediates tonic impairment of learning. In response to stressful stimuli or after local injection of high CRF doses, CRFR2 mediates anxiety. Effects requiring CRFR2 can be blocked specifically by the recently developed peptidic antagonist antisauvagine-30.

L2 ANSWER 54 OF 66 MEDLINE on STN DUPLICATE 25

ACCESSION NUMBER: 1999355035 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10428081
TITLE: The ligand-selective domains of corticotropin-releasing factor type 1 and type 2 receptor reside in different extracellular domains: generation of chimeric receptors with a novel ligand-selective profile.
AUTHOR: Dautzenberg F M; Kilpatrick G J; Wille S; Hauger R L
CORPORATE SOURCE: Preclinical Research, Pharma Division, F. Hoffmann-La Roche Ltd., Basel, Switzerland.
CONTRACT NUMBER: MH20914-14 (NIMH)
SOURCE: Journal of neurochemistry, (1999 Aug) Vol. 73, No. 2, pp. 821-9.
Journal code: 2985190R. ISSN: 0022-3042.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199908
ENTRY DATE: Entered STN: 19990827
Last Updated on STN: 19990827
Entered Medline: 19990813

AB The nonselective human corticotropin-releasing factor (hCRF) receptor 1 (hCRFR1) and the ligand-selective Xenopus CRFR1 (xCRFR1), xCRFR2, and hCRFR2alpha were compared. To understand the interactions of hCRF, ovine CRF (oCRF), rat urocortin (rUcn), and sauvagine, ligands with different affinities for type 1 and type 2 **CRFRs**, chimeric and mutant receptors of hCRFR1, xCRFR1, hCRFR2alpha, and xCRFR2 were constructed. In cyclic AMP stimulation and CRF-binding assays, it was established that different extracellular regions of CRFR1 and CRFR2 conferred their ligand selectivities. The ligand selectivity of xCRFR1 resided in five N-terminal amino acids, whereas the N-terminus of both CRFR2 proteins did not contribute to their ligand selectivities. Chimeric receptors in which the first extracellular domain of hCRFR1 replaced that of hCRFR2alpha or xCRFR2 showed a similar pharmacological profile to the two parental CRFR2 molecules. Chimeric receptors carrying the N-terminal domain of xCRFR1 linked to hCRFR2alpha or xCRFR2 displayed a novel pharmacological profile. hCRF, rUcn, and sauvagine were bound with high affinity, whereas oCRF was bound with low affinity. Furthermore, when three or five residues of xCRFR1 (Gln76, Gly81, Val83, His88, Leu89; or Gln76, Gly81, Val83) were introduced into receptor chimeras carrying the N-terminus of hCRFR1 linked to xCRFR2, the same novel pharmacology was observed. These data indicate

a compensation mechanism of two differentially selecting regions located in different domains of both xCRFR1 and CRFR2.

L2 ANSWER 55 OF 66 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN DUPLICATE 26

ACCESSION NUMBER: 1999:492964 BIOSIS
DOCUMENT NUMBER: PREV199900492964
TITLE: Transfer of 238U, 226Ra and 210Pb from slag-contaminated soils to vegetables under field conditions.
AUTHOR(S): Bunzl, K. [Reprint author]; Trautmannsheimer, M.
CORPORATE SOURCE: GSF-National Research Center for Environment and Health, Institute of Radiation Protection, 85764, Neuherberg, Germany
SOURCE: Science of the Total Environment, (July 1, 1999) Vol. 231, No. 2-3, pp. 91-99. print.
CODEN: STENDL. ISSN: 0048-9697.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 16 Nov 1999
Last Updated on STN: 16 Nov 1999

AB The uptake of 238U, 226Ra and 210Pb by beans (dwarf bean Modus), kohlrabi (brassica oleracea var. gongylodes), mangold (beta vulgaris var. macrorhiza), lettuce (American gathering brown), carrots (Rotin, Sperlings's), celery, and radish from a control soil and from soil/slag mixtures was investigated in field experiments. The observed concentration ratios (CR) suggest that 226Ra in the slag/soil mixtures is less available for root uptake than 226Ra in the control soil. This is especially evident for beans, mangold and carrots, and less conspicuous for lettuce. A similar behaviour was observed for the uptake of 238U by celery and radish, but not in the case of lettuce. In this latter case, the CR was even higher when this vegetable was grown on a coal slag contaminated soil when compared to the pure control soil. It is shown that the root uptake of a natural radionuclide from a slag contaminated soil in excess to that from an uncontaminated soil can be characterized best by defining a fractional concentration ratio (**CRfr**). In this way, all above results are quantified with respect to the root uptake of the radionuclides from the slag fraction in the soils. Because **CRfr** is not affected by atmospheric contributions to the plant activity, this quantity can be also used to obtain the root uptake of 210Pb by vegetation which results only from the slag fraction in the soil. By comparing the **CRfr**-values observed for different radionuclides, it is shown that e.g. the amount of 210Pb root-uptake by lettuce from the slags is similar to that of 238U and 226Ra.

L2 ANSWER 56 OF 66 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 1999:161704 BIOSIS
DOCUMENT NUMBER: PREV199900161704
TITLE: Neuroendocrine and immune dysfunctions in CRFR1-deficient mice.
AUTHOR(S): Lee, Kuo-Fen [Reprint author]; Aubry, Jean-Michel [Reprint author]; Dellu, Francoise; Bilezikjian, Louise M. [Reprint author]; Turnbull, Andrew [Reprint author]; Gold, Lisa; Chen, Ruoping [Reprint author]; Marchuk, Yelena [Reprint author]; Hauser, Chris [Reprint author]; Sawchenko, Paul E.; Koob, George; Vale, Wylie [Reprint author]; Smith, George W. [Reprint author]
CORPORATE SOURCE: Clayton Foundation Lab., Peptide Biol., Salk Inst., La Jolla, CA 92037, USA
SOURCE: Oomura, Y. [Editor]; Hori, T. [Editor]. (1998) pp. 83-96. Taniguchi Symposia on Brain Sciences; Brain and biodefence. print.
Publisher: Japan Scientific Societies Press, 2-10 Hongo, 6-chome, Bunkyo-ku, Tokyo, Japan; S. Karger AG, New York,

New York, USA. Series: Taniguchi Symposia on Brain Sciences.
Meeting Info.: Twenty-First International Taniguchi Symposium on Brain Sciences with the main theme of Brain and the Biodefense System. Kyoto, Japan. January 19-22, 1998. Taniguchi Foundation and the Executive Committee.
ISBN: 3-8055-6764-2.
DOCUMENT TYPE: Book; (Book Chapter)
Conference; (Meeting)
Conference; (Meeting Paper)
LANGUAGE: English
ENTRY DATE: Entered STN: 16 Apr 1999
Last Updated on STN: 16 Apr 1999

L2 ANSWER 57 OF 66 MEDLINE on STN DUPLICATE 27
ACCESSION NUMBER: 1999080001 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9860957
TITLE: Structural requirements for peptidic antagonists of the corticotropin-releasing factor receptor (CRFR): development of CRFR2beta-selective antisauvagine-30.
AUTHOR: Ruhmann A; Bonk I; Lin C R; Rosenfeld M G; Spiess J
CORPORATE SOURCE: Department of Molecular Neuroendocrinology, Max Planck Institute for Experimental Medicine, Hermann-Rein-Strasse 3, D-37075 Gottingen, Germany.
SOURCE: Proceedings of the National Academy of Sciences of the United States of America, (1998 Dec 22) Vol. 95, No. 26, pp. 15264-9.
Journal code: 7505876. ISSN: 0027-8424.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199901
ENTRY DATE: Entered STN: 19990209
Last Updated on STN: 19990209
Entered Medline: 19990128

AB Different truncated and conformationally constrained analogs of corticotropin-releasing factor (CRF) were synthesized on the basis of the amino acid sequences of human/rat CRF (h/rCRF), ovine CRF (oCRF), rat urocortin (rUcn), or sauvagine (Svg) and tested for their ability to displace [125I-Tyr0]oCRF or [125I-Tyr0]Svg from membrane homogenates of human embryonic kidney (HEK) 293 cells stably transfected with cDNA coding for rat CRF receptor, type 1 (rCRFR1), or mouse CRF receptor, type 2beta (mCRFR2beta). Furthermore, the potency of CRF antagonists to inhibit oCRF- or Svg-stimulated cAMP production of transfected HEK 293 cells expressing either rCRFR1 (HEK-rCRFR1 cells) or mCRFR2beta (HEK-mCRFR2beta cells) was determined. In comparison with astressin, which exhibited a similar affinity to rCRFR1 (Kd = 5.7 +/- 1.6 nM) and mCRFR2beta (Kd = 4.0 +/- 2.3 nM), [DPhe11,His12]Svg(11-40), [DLeu11]Svg(11-40), [DPhe11]Svg(11-40), and Svg(11-40) bound, respectively, with a 110-, 80-, 68-, and 54-fold higher affinity to mCRFR2beta than to rCRFR1. The truncated analogs of rUcn displayed modest preference (2- to 7-fold) for binding to mCRFR2beta. In agreement with the results of these binding experiments, [DPhe11, His12]Svg(11-40), named antisauvagine-30, was the most potent and selective ligand to suppress agonist-induced adenylate cyclase activity in HEK cells expressing mCRFR2beta.

L2 ANSWER 58 OF 66 MEDLINE on STN DUPLICATE 28
ACCESSION NUMBER: 1998391555 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9725716
TITLE: Stress-induced changes of gene expression in the paraventricular nucleus are enhanced in spontaneously hypertensive rats.
AUTHOR: Imaki T; Naruse M; Harada S; Chikada N; Nakajima K;

Yoshimoto T; Demura H
CORPORATE SOURCE: Department of Medicine, Institute of Clinical Endocrinology, Tokyo Women's Medical College, Japan.
SOURCE: Journal of neuroendocrinology, (1998 Aug) Vol. 10, No. 8, pp. 635-43.
Journal code: 8913461. ISSN: 0953-8194.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199811
ENTRY DATE: Entered STN: 19990106
Last Updated on STN: 19990106
Entered Medline: 19981110

AB Heightened hypothalamic-pituitary-adrenal (HPA) axis responses have been implicated in hypertension in the spontaneously hypertensive rat (SHR), but the exact mechanisms involved are poorly understood. To determine changes in gene expression in SHR in the paraventricular nucleus (PVN), stress-induced accumulation of CRF, CRF type 1 receptor (CRFR-1) genes, and immediate-early genes were examined using in situ hybridization in young (5 weeks old) and adult (12 weeks old) stroke-prone SHR (SHRSP), compared with normotensive Wistar Kyoto (WKY) rats. Restraint stress-induced accumulation of c-fos, jun B, and NGFI-B mRNA, and CRF hnRNA in the PVN was significantly higher in young and adult SHRSP than in WKY rats at 30 min, except for c-fos in young rats. CRFR-1 mRNA expression in the PVN was also significantly higher in adult SHRSP than in WKY rats at 120 min after stress onset. CRF mRNA was increased in response to stress in young SHRSP. The basal CRF mRNA level in the PVN was significantly lower in adult SHRSP than in WKY rats. Young SHRSP exhibit greater ACTH responses to stress without significant changes in plasma corticosterone concentrations. The adult SHRSP exhibited lower baseline concentrations of corticosterone and similar corticosterone response to stress with enhanced secretion of ACTH. Overall, these results demonstrated that stress-induced activation of immediate early genes and CRF gene transcription in the PVN, and ACTH secretion is enhanced in early hypertensive, young, and adult SHRSP, suggesting that they are probably not the result of chronic alterations in blood pressure. The abnormal hypothalamic-pituitary response to stress thus appears to be related to the development of hypertension.

L2 ANSWER 59 OF 66 MEDLINE on STN DUPLICATE 29
ACCESSION NUMBER: 1999037955 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9822161
TITLE: Characterization of native corticotropin-releasing factor receptor type 1 (CRFR1) in the rat and mouse central nervous system.
AUTHOR: Radulovic J; Sydow S; Spiess J
CORPORATE SOURCE: Department of Molecular Neuroendocrinology, Max Planck Institute for Experimental Medicine, Goettingen, Germany.
SOURCE: Journal of neuroscience research, (1998 Nov 15) Vol. 54, No. 4, pp. 507-21.
Journal code: 7600111. ISSN: 0360-4012.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199901
ENTRY DATE: Entered STN: 19990202
Last Updated on STN: 19990202
Entered Medline: 19990119

AB Corticotropin releasing factor (CRF), the most important regulator of various responses to stress, acts through CRF receptors (CRFR). For their characterization in brain tissue of Sprague-Dawley rats and C57BL/6J mice, a recently described polyclonal antibody directed against

the N-terminus of rat CRFR1 (rCRFR1) was used. The molecular weights of rat and mouse brain receptors were determined by Western blot analysis to be 80,000-76,000 and 83,000-79,000, respectively, whereas molecular weights of 72,000-59,000 were observed for CRFR1 from rat and mouse pituitary. Immunohistochemical analysis was performed with brain sections of naive rats and mice. Strong CRFR1 staining was detected in the cortex, cerebellum, mesencephalon and pons of both species, whereas weak staining was observed in amygdala and hippocampus. The striatum did not show immunoreactivity. The density of immunostaining was significantly lower in murine than in rat cortex. In contrast, in the pons and mesencephalon of mice, higher density of immunostaining was observed than in the same brain structures of rats. On the basis of the observed differences, it is suggested that CRFR1 is differentially processed in rats and mice. In addition, the density of CRFR1 staining differed between both species.

L2 ANSWER 60 OF 66 MEDLINE on STN DUPLICATE 30
ACCESSION NUMBER: 97322103 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9178757
TITLE: Localization of ligand-binding domains of human corticotropin-releasing factor receptor: a chimeric receptor approach.
AUTHOR: Liaw C W; Grigoriadis D E; Lovenberg T W; De Souza E B; Maki R A
CORPORATE SOURCE: Department of Molecular Neurobiology, Neurocrine Biosciences, Inc., San Diego, California 92121, USA.
CONTRACT NUMBER: R43 NS34203 (NINDS)
SOURCE: Molecular endocrinology (Baltimore, Md.), (1997 Jun) Vol. 11, No. 7, pp. 980-5.
Journal code: 8801431. ISSN: 0888-8809.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199709
ENTRY DATE: Entered STN: 19971008
Last Updated on STN: 19971008
Entered Medline: 19970923

AB Two CRF receptors, CRFR1 and CRFR2, have recently been cloned and characterized. CRFR1 shares 70% sequence identity with CRFR2, yet has much higher affinity for rat/human CRF (r/hCRF) than CRFR2. As a first step toward understanding the interactions between rat/human CRF and its receptor, the regions that are involved in receptor-ligand binding and/or receptor activation were determined by using chimeric receptor constructs of the two human CRFR subtypes, CRFR1 and CRFR2, followed by generating point mutations of the receptor. The EC50 values in stimulation of intracellular cAMP of the chimeric and mutant receptors for the peptide ligand were determined using a cAMP-dependent reporter system. Three regions of the receptor were found to be important for optimal binding of r/hCRF and/or receptor activation. The first region was mapped to the junction of the third extracellular domain and the fifth transmembrane domain; substitution of three amino acids of CRFR1 in this region (Val266, Tyr267, and Thr268) by the corresponding CRFR2 amino acids (Asp266, Leu267, and Val268) increased the EC50 value by approximately 10-fold. The other two regions were localized to the second extracellular domain of the CRFR1 involving amino acids 175-178 and His189 residue. Substitutions in these two regions each increased the EC50 value for r/hCRF by approximately 7- to 8-fold only in the presence of the amino acid 266-268 mutation involving the first region, suggesting that their roles in peptide ligand binding might be secondary.

L2 ANSWER 61 OF 66 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1998:361908 CAPLUS
DOCUMENT NUMBER: 129:118023
TITLE: Regulation of gene expression in the hypothalamus by

stress: corticotropin-releasing factor (CRF), CRF receptor and oncogene
AUTHOR(S): Imaki, Toshihiro
CORPORATE SOURCE: Tokyo Women's Medical College, Japan
SOURCE: Undo Seikagaku (1997), 9, 3-12
CODEN: UNSEFC; ISSN: 0915-4515
PUBLISHER: Minsei Kagaku Kyokai
DOCUMENT TYPE: Journal
LANGUAGE: Japanese

AB The authors examined the regulation CRF, CRF receptor gene and oncogene expression in the hypothalamic paraventricular nucleus (PVN), which plays an important role in stress response. CRF transcription was analyzed by intron-directed in situ hybridization to detect CRF hnRNA. CRF hnRNA expression was rapidly induced (within 5 min.) following stress, suggesting that CRF gene transcription increases in response to stress. Since none of the oncogene transcripts including c-fos, jun B, NGFI-B increase before than of CRF hnRNA, it is unlikely that the products of these oncogenes mediate activation of CRF gene transcription. CRF type 1 receptor (CRFR-1) mRNA expression was increased in the PVN after, icv injection of CRF, and methyrapone administration. Thus, CRF may stimulate its own biosynthesis and/or release within the PVN by increasing CRFR-1 expression. In contrast, centrally-given urocortin, an intrinsic ligand for CRFR-2, decreased CRF mRNA in the PVN, whereas α-helical CRF, CRFR-2 antagonist, increased CRF mRNA levels. Therefore, urocortin-CRFR-2 may possibly inhibit CRF synthesis in the PVN.

L2 ANSWER 62 OF 66 MEDLINE on STN DUPLICATE 31
ACCESSION NUMBER: 96278921 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8662941
TITLE: The genomic structure of the rat corticotropin releasing factor receptor. A member of the class II G protein-coupled receptors.
AUTHOR: Tsai-Morris C H; Buczko E; Geng Y; Gamboa-Pinto A; Dufau M L
CORPORATE SOURCE: Section on Molecular Endocrinology, Endocrinology and Reproduction Research Branch, NICHD, National Institutes of Health, Bethesda, Maryland 20892-4510, USA.
SOURCE: The Journal of biological chemistry, (1996 Jun 14) Vol. 271, No. 24, pp. 14519-25.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-U53486; GENBANK-U53487; GENBANK-U53488; GENBANK-U53489; GENBANK-U53490; GENBANK-U53491; GENBANK-U53492; GENBANK-U53493; GENBANK-U53494; GENBANK-U53495; GENBANK-U53496; GENBANK-U53497; GENBANK-U53498; GENBANK-U53499; GENBANK-U53500
ENTRY MONTH: 199608
ENTRY DATE: Entered STN: 19960828
Last Updated on STN: 19960828
Entered Medline: 19960820

AB Isolation and structural characterization of the rat corticotropin releasing factor receptor (CRFR) gene was performed to determine the exon/intron organization of the coding region and the potential for splice variants. The CRFR gene contains 13 exons and 12 introns, and the positions of the exon/intron junctions are similar to those of other Class II G protein-coupled receptor genes including the parathyroid hormone and glucagon receptors. The promoter resides within 593 base pairs of the initiation codon and the major transcriptional start site at nucleotide -238. This domain does not possess a TATA box but contains multiple Sp1 and AP-2 sites upstream and downstream of the major

transcriptional start site. Intron junctions were identified in the extracellular, transmembrane (TM), and cytoplasmic (C) domains of the CRFR, giving the potential for differential signal transduction by splice variants. CRFR cDNAs derived from rat Leydig cell mRNA included the pituitary Form A, which spans exons 1-13, and two splice variants with deletion of exon 3 or exons 7, 11, and 12. An evolutionary link between the intronless TM/C module of the glycoprotein hormone receptors and the intron-containing TM/C module of the CRFR is suggested by the common position of the luteinizing hormone receptor Form D alternate acceptor splice site and the CRFR intron 12.

L2 ANSWER 63 OF 66 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1992:652193 CAPLUS
DOCUMENT NUMBER: 117:252193
TITLE: Large deformation of polycarbonate near the glass transition temperature
AUTHOR(S): Inoue, Tadashi; Okamoto, Hirotaka; Osaki, Kunihiro
CORPORATE SOURCE: Inst. Chem. Res., Kyoto Univ., Uji, 611, Japan
SOURCE: Macromolecules (1992), 25(25), 7069-70
CODEN: MAMOBX; ISSN: 0024-9297
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The stress, f, and the birefringence, Δn, of bisphenol A polycarbonate were measured at 151° during an elongation at constant speed (initial rate of elongation, .ovrhdot.e0 = 1.0 + 10-4 - 7.4 + 10-3 s-1). The data were analyzed with the modified stress-optical rule, in which each of f and Δn was assumed to be a sum of 2 terms and the stress-optical rule was assumed valid sep. for each set of the components: f = fR + fG and Δn = CRfR + CGfG, where CR and CG were material consts. The R component of stress, fR, increased monotonously with the elongation ratio, λ. The G component, fG, originating high glassy modulus, showed complicated λ dependence: at .ovrhdot.e0 = 1.0 + 10-4 - 1.0 + 10-3 s-1, but it vanished over a certain range of λ, where the ordinary stress-optical rule held well. The results were compared with those for a polystyrene, and quant. differences were found in the λ dependence of the G components.

L2 ANSWER 64 OF 66 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1992:408856 CAPLUS
DOCUMENT NUMBER: 117:8856
TITLE: Birefringence of amorphous polymers. 4. Large deformation of polystyrene near its glass transition temperature
AUTHOR(S): Okamoto, Hirotaka; Inoue, Tadashi; Osaki, Kunihiro
CORPORATE SOURCE: Inst. Chem. Res., Kyoto Univ., Uji, 611, Japan
SOURCE: Macromolecules (1992), 25(13), 3413-15
CODEN: MAMOBX; ISSN: 0024-9297
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The stress, f, and the birefringence, Δn, were measured for polystyrene at 98° under large deformations after an instantaneous elongation (0.6-2%) and during an elongation at constant speed (initial rate of elongation = 5.5 + 10-4 - 2 + 10-1 s-1). The stress-optical rule was not valid. The data were analyzed in terms of the modified stress-optical rule, in which each f and Δn was assumed to be a sum of 2 terms and the stress-optical rule was assumed valid sep. for each set of components: f = fR + fG and Δn = CRfR + CGfG, where CR and CG were material consts. The slowly relaxing component of Young's relaxation modulus, ER = fR/e, was independent of the strain, e, while the rapidly relaxing component, EG = fG/e, decreased with strain. At a constant speed of elongation, 1 component of stress, fR, increased with time and was close to that evaluated from ER with linear viscoelasticity theory. The other component, fG, exhibited a marked overshoot and then

decreased with time. This component was much smaller than that derived from EG through a linear viscoelasticity relation. The yield phenomenon at a constant speed of elongation was due to the behavior of the G-component, and the slow increase of stress at long times was due to the increase of the R-component, presumably associated with the deformation of polymer segments.

L2 ANSWER 65 OF 66 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 1991:408651 BIOSIS
DOCUMENT NUMBER: PREV199192075616; BA92:75616
TITLE: MATURATION AND FUNCTION OF CYSTIC FIBROSIS TRANSMEMBRANE CONDUCTANCE REGULATOR VARIANTS BEARING MUTATIONS IN PUTATIVE NUCLEOTIDE-BINDING DOMAINS 1 AND 2.
AUTHOR(S): GREGORY R J [Reprint author]; RICH D P; CHENG S H; SOUZA D W; PAUL S; MANAVALAN P; ANDERSON M P; WELSH M J; SMITH A E
CORPORATE SOURCE: GENZYME CORP, ONE MOUNTAIN RD, FRAMINGHAM, MASS 01701, USA
SOURCE: Molecular and Cellular Biology, (1991) Vol. 11, No. 8, pp. 3886-3893.
CODEN: MCEBD4. ISSN: 0270-7306.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH
ENTRY DATE: Entered STN: 11 Sep 1991
Last Updated on STN: 11 Sep 1991

AB One feature of the mutations thus far found to be associated with the disease cystic fibrosis (CF) is that many of them are clustered within the first nucleotide-binding domain (NBD) of the CF transmembrane conductance regulator (CFTR). We sought to discover the molecular basis for this clustering by introducing into the two NBDs of CFTR mutations either mimicking amino acid changes associated with CF or altering residues within highly conserved motifs. Synthesis and maturation of the mutant CFTR were studied by transient expression in COS cells. The ability of the altered proteins to generate cyclic AMP-stimulated anion eflux was assessed by using 6-methoxy-N-(sulfopropyl) quinolinium (SPQ) fluorescence measurements in HeLa cells expressing mutated plasmids. The results show that (i) all CF-associated mutants, with one exception, lack functional activity as measured in the SPQ assay, (ii) mutations in NBD1 are more sensitive to the effects of the same amino acid change than are the corresponding mutations in NBD2, (iii) cells transfected with plasmids bearing CF-associated mutations commonly but not exclusively lack mature CFTR, (iv) NBD mutants lacking mature CFTR fail to activate Cl- channels, and (v) the glycosylation of CFTR, per se, is not required for CFTR function. We reason that the structure of NBD1 itself or of the surrounding domain renders it particularly sensitive to mutational changes. As a result, most NBD1 mutants, but only a few NBD2 mutants, fail to mature or lack functional activity. These findings are consistent with the observed uneven distribution of **CRFR** missense mutations between NBD1 and NBD2 or CF patients.

L2 ANSWER 66 OF 66 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1956:64064 CAPLUS
DOCUMENT NUMBER: 50:64064
ORIGINAL REFERENCE NO.: 50:11916f-g
TITLE: The effect of titanium on the intergranular corrosion of stainless steel containing eighteen percent chromium and nine percent nickel
AUTHOR(S): Cihal, Vladimir; Pospisil, Rudolf
CORPORATE SOURCE: Vyzkumny Ustav Ochrany materialu, Prague
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AB In 18/8 steels stabilized with Ti, Ti carbide begins to dissolve at higher

temps. At 650° mainly (CrFr)23C6 carbides precipitate from the oversatd. solid solution, and the steel is susceptible to intergranular corrosion. The effect of Ti carbide is discussed in detail.